Neurologic examination – gait and postural reactions

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Most neurologic deficits are best seen during slow walk and at turns. Remember to observe the patient from all directions. If it is difficult to determine an abnormality, evaluate the horse on a slope. Examine the patient loos in a confined area such a paddock may also be of value.

Pattern recognition in is crucial in evaluating gait disorders. These patterns have five components: two components with paresis (weakness) and three components of ataxia.

Paresis:

Paresis is weakness but it also includes generation of the gait or the ability to support weight. Paresis can be caused by lower motor neuron (LMN) and upper motor neuron (UMN) deficiencies.

LMN deficiencies:

Difficulties in supporting weight and varies from short stride to complete inability to support weight. Typical signs of LMN deficiencies are:

- Hypotonicity or atonicity
- Hyporeflexia
- Flaccidity
- Denervation atrophy
- Fasciculations

UMN deficiencies

Paresis in UMN causes:

- Hypertonicity
- Hyperreflexia
- Spasticity
- Muscle weakness

UMN paresis is clinically characterized by:

- Delay in protraction of the swing phase
- Usually longer stride
- Stiffness and spasticity (in the stride)
- Hoof slap when the hoof hits ground

Ataxia:

Ataxia is a synonym for incoordination. There are three types of ataxia:

- 1. General proprioceptive ataxia
- 2. Vestibular ataxia (special proprioception)
- 3. Cerebellar ataxia

Cerebellar ataxia is uncommon. The horse usually has intension tremor and a hypermetric gait. Vestibular ataxia is reflects loss of orientation involving the eyes, neck, trunk and limbs. This result in loss of balance and the horse is usually leaning, drifting or falling to one side.

General proprioceptive (GP) ataxia is characterized by lack of information where the neck, trunk and limbs are in space. Without the GP information to the CNS the onset of protraction is delayed, the stride might be longer and the limb may swing to the side (abducts) or under the body (adduct) – especially during circling. Observe that these signs overlap with those caused by dysfunction of the UMN.

Clinical signs in horses with general proprioceptive ataxia:

- Base wide stance (or stand with legs in abnormal positions)
- Delayed protraction longer stride lenght (flexorparesis)
- Hypometria (increased extensoraktivitet)
- Hypermetria (spinocerebellar tracts: GP information to cerebellum)
- Float (frontlegs)
- Toe dragging
- Circling: (pivot, circumduction, crossing over, toe dragging)
- Tail pull: paresis

Postural reactions:

In horses, testing for postural reaction cannot be performed as for small animals. The horse is usually evaluated during head elevation (walking with the head in a high position), circling and backing. Circling should be performed by the horse owner as well as by the examiner. Walk the horse in a tight circle 8 – 10 times in each direction. The leader should always be in the center of the circle. Never circle the horse with the leader on the outer side. Common signs for UMN deficiencies and GP ataxia during circling are pivot on the inside limb, circumduction, crossing over and toe dragging on the outer leg.

Sway the standing horse from side to side and evaluate its muscle strength. Sway the horse when it is walking by pulling its tail towards you and then releasing the tension. The degree of resistance will help you to assess the horse's strength.